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# Treadmill exercise duration and dyspnea recovery time in chronic obstructive pulmonary disease: effects of oxygen breathing and repeated testing

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Oxygen supplementation is known to improve exercise capacity in patients with chronic obstructive pulmonary disease (COPD). Although some COPD patients use oxygen after exercise to relieve dyspnea, the effect of oxygen during recovery from exercise is not clearly understood. Exercise duration and dyspnea recovery time were studied in 18 patients with stable COPD. Patients exercised at a constant submaximal work rate on a treadmill ergometer until they no longer wished to continue. Oxygen, room air and compressed air were randomly administered in three consecutive post-exercise recovery periods. Dyspnea was scored on a 100 mm visual analog scale at 30 s intervals until return to baseline. An additional 20 minute post-recovery resting period was allowed between each test. No significant differences were found in dyspnea recovery time breathing oxygen (271 s), room air (290 s) or compressed air (311 s). When the groups were sorted by sequence of testing, there was a highly significant increase in recovery time (208 s, 307 s and 358 s for the first, second and third tests;  $P < 0.005$ ) and a non-statistically significant decrease in exercise duration (89 s, 79 s and 76 s). Post-exercise oxygen supplementation had no effect on dyspnea recovery time in these COPD patients. Repeated bouts of exercise increased dyspnea recovery time and tended to decrease exercise duration. These findings suggest that, despite recovery of symptoms, physiological recovery from prior exercise is incomplete.

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## Introduction

Exercise performance is impaired in patients with chronic obstructive pulmonary disease (COPD) by reduced ventilatory capacity (1,2), ventilatory inefficiency (high  $V_D/V_T$ ), and physical deconditioning leading to premature lactic acidosis (3). Administration of supplemental oxygen improves exercise tolerance and reduces breathlessness in COPD patients (4); this is achieved by a reduction in ventilatory requirement for the same work load (5). Pre-exercise administration of oxygen was shown to reduce breathlessness for short exercise bouts in these patients (4).

Oxygen is commonly used by COPD patients to relieve dyspnea during recovery from exercise. Despite the beneficial effect of oxygen inhalation on exercise performance of COPD patients, its effect on exercise recovery is unclear. In one study (6), 19 COPD patients inhaled oxygen, room air or compressed air during recovery from maximal exercise using a 31 cm step. While 11 felt relief of breathlessness as shown by visual analog scales (VASs),

respiratory rate and heart rate were not affected by the gas inhaled. Furthermore, these results were not reproducible in later tests on the same patients.

The present study was designed to ascertain the effect of oxygen breathing and repeated bouts of maximal treadmill exercise on dyspnea recovery time in COPD patients. We chose treadmill walking as an exercise that is closely related to everyday exercise activities. Our hypothesis was that repeated bouts of exercise would tend to prolong recovery time breathing room air and that breathing supplemental oxygen would shorten recovery time.

## Materials and Methods

### STUDY DESIGN

This was a randomized, cross-over, single-blind trial to compare compressed oxygen, compressed air and room air on recovery from exercise-induced dyspnea. The subjects were unaware of the type of compressed gas being administered. In order to minimise unconscious bias, the supervising technician used standard statements of encouragement for each test. Random numbers were generated from a random number table to establish the sequence of gas administered during recovery from each of the three tests.

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## SUBJECTS

Eighteen COPD patients with severe airflow obstruction ( $FEV_1 < 40\%$  predicted) were recruited (14 male, four female). They were clinically stable without evidence of a significant exacerbation of their airflow obstruction within 3 months prior to participation in the study. Exclusion criteria were the presence of symptomatic ischemic heart disease, asthma with reversibility of  $FEV_1 > 15\%$  after inhalation of 400  $\mu\text{g}$  of salbutamol or exercise tolerance limited by other disabling disease. The study received Human Subject Protection Committee approval and participants gave written informed consent.

## METHODS

All patients had lung volumes and forced expiratory flows measured by spirometry. Functional residual capacity (FRC) was measured using the helium dilution technique. The transfer factor for carbon monoxide (TLCO) was determined by the single-breath technique. Arterial blood gases were measured at rest while room air was being breathed. The subjects were familiarized with walking on a treadmill and with the use of a 100 mm VAS.

The exercise tests were performed on a treadmill ergometer with 0% grade. The treadmill speed was adjusted immediately before the first test to suit each individual subject, based on comfort and preference. The same treadmill grade and speed were then used for all subsequent tests. On the day of testing, three exercise bouts were performed with the subject breathing room air. During the recovery periods the subject breathed room air, compressed air or compressed oxygen as randomly assigned. The chosen recovery time of 20 min allowed for complete recovery of symptoms after each study. Compressed air and oxygen were delivered through a low-flow face mask (M/C mask). The gas flow was fixed at  $10 \text{ l min}^{-1}$  for both compressed air and oxygen. This type of mask delivers a fractional inspired oxygen concentration ( $FiO_2$ ) of approximately 40%. The experimental conditions were intended to reflect the clinical circumstances under which patients take supplemental oxygen, accepting that an absolutely constant  $FiO_2$  could not be guaranteed during oxygen administration. The VAS for breathlessness was completed immediately when exercise stopped and at 30 s intervals until the score was less than, or within 5 mm of, the baseline. A 20 min rest period after complete subjective recovery followed each exercise bout.

## STATISTICAL ANALYSIS

Anthropometric, pulmonary function and arterial blood gas data were expressed as mean  $\pm$  SD. Data were entered into a microcomputer data base and analysed with a commercial statistical package. The results for the different testing conditions were compared using two-way analysis of variance for repeated measures. Differences between means were assessed using Student's *t* test for paired data with Bonferroni corrections whenever appropriate. Values of  $P < 0.05$  were considered significant.

TABLE 1. Pulmonary function and arterial blood gas analysis in the 18 subjects

Age (years)	63.3 $\pm$ 5.8
$FEV_1$ (l)	0.87 $\pm$ 0.44
$FEV_1$ (%)	27 $\pm$ 13
FVC (l)	1.87 $\pm$ 0.63
TLC (l)	5.49 $\pm$ 1.38
RV/TLC (%)	55.5 $\pm$ 7.7
TLCO ( $\text{ml min}^{-1} \text{ mmHg}^{-1}$ )	10.3 $\pm$ 5.8
$PaO_2$ (kPa)	6.8 $\pm$ 1.5
$PaCO_2$ (kPa)	6.1 $\pm$ 1.1

Values are mean  $\pm$  standard deviation.

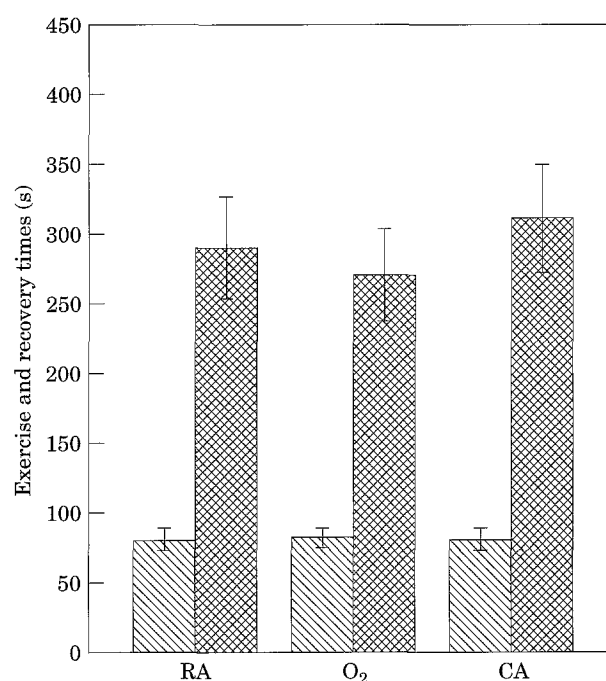


FIG. 1. Exercise (▨) and recovery (▩) times sorted by gas breathed: room air (RA), compressed air (CA) or oxygen ( $O_2$ ).

## Results

Eighteen patients completed the study. Table 1 shows age, baseline pulmonary function data and arterial blood gases for the group. They had moderately severe airflow obstruction, hyperinflation, impaired gas transfer, hypoxemia and hypercapnia.

When the exercise tests were grouped according to the gas breathed during recovery, no significant difference was observed in exercise duration or dyspnea recovery time (Fig. 1). For breathing room air, exercise duration was  $81 \pm 8$  s and dyspnea recovery time was  $290 \pm 37$  s. For breathing oxygen, exercise duration was  $83 \pm 7$  s and dyspnea recovery time was  $271 \pm 33$  s. For breathing compressed air, exercise duration was  $81 \pm 8$  s and recovery time was  $311 \pm 39$  s.

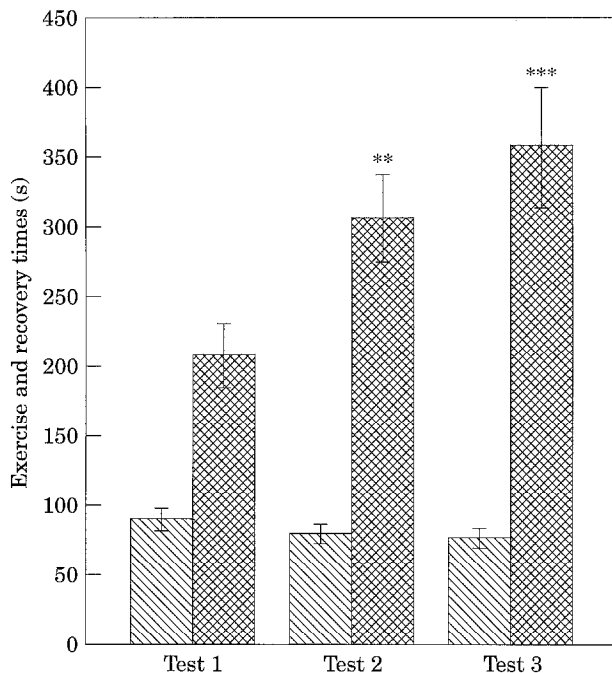


FIG. 2. Exercise (▨) and recovery (▩) times sorted by test sequence. Statistical significance: \* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$  (paired  $t$  test).

When the studies were analysed according to the sequence in which they were performed, a highly significant prolongation of dyspnea recovery time was observed with each consecutive test while the actual duration of exercise became shorter (Fig. 2). On the first test, the exercise duration was  $89 \pm 8$  s and the dyspnea recovery time was  $208 \pm 23$  s. On the second test, exercise duration was  $79 \pm 7$  s and dyspnea recovery time was  $307 \pm 31$  s ( $P < 0.01$ ). On the third test, exercise duration was  $76 \pm 7$  s and dyspnea recovery time was  $358 \pm 43$  s ( $P < 0.001$ ). Values are means  $\pm$  SEM and statistical comparisons are with the first test.

## Discussion

Unexpectedly, we found no effect of post-exercise oxygen breathing on dyspnea recovery time in our COPD patients. Previous exercising significantly prolonged recovery time and tended to shorten exercise time.

In the analysis comparing different gases inhaled during recovery, the duration of exercise was consistent between subgroups. Naturally, we would not expect oxygen administration during the recovery period to influence the duration of the preceding exercise bout; rather this shows that a bias due to exercise repetition was unlikely to be affecting our results. We believe the number of subjects participating in this study was sufficient to overcome a type II statistical error. However, since the influence of the sequence of tests is so great, this might have confounded a much smaller effect from the gas inhaled during recovery.

Nevertheless, if such an effect existed, our study suggests that it is too small to have clinical relevance.

Our results contrast with those of a previous study which reported shorter dyspnea recovery time in COPD subjects with oxygen supplementation (6). In this previous study, however, the investigators were unable to demonstrate a consistent response when the step-exercise protocol was repeated in some of their subjects. In our study, repetition of treadmill walking exercise led to prolongation of recovery time. Exercise time was reduced by 14.6%, when comparing the first and the last exercise bout; however, this was not statistically significant using repeated-measures analysis of variance. The increase in recovery time was much greater: 47.5% following the second exercise bout ( $P < 0.01$ ) and 72% following the third ( $P < 0.001$ ). Evans *et al.* (6) also reported an increased recovery time but no decrease in exercise duration for their step-exercise protocol.

Repetition of heavy exercise in healthy subjects does not affect maximum oxygen uptake but reduces endurance time when the tests are separated by less than 30 min (7). In COPD patients  $\dot{V}O_2$  kinetics is slowed, causing a greater oxygen debt (8). Post-exercise lactic acid levels are likely to be higher, resulting in prolonged elevation of carbon dioxide output and  $\dot{V}_E$ . Hence it is likely that the patients we studied had not achieved full metabolic recovery before starting the next exercise bout. Nevertheless, breathlessness scores were observed to have returned to less than or within 5 mm of baseline during each recovery period. Since it is known that the VAS score correlates with  $\dot{V}_E$  both during exercise and recovery (9), it is likely that  $\dot{V}_E$  in our subjects was close to baseline before the next exercise bout; however, we did not measure  $\dot{V}_E$  directly.

In normal subjects, dyspnea during exercise correlates with minute ventilation ( $\dot{V}_E$ ). Also hypoxemia and hypercapnia have been shown to contribute to the sensation of dyspnea and could do so by stimulating an increase in ventilation rather than as a direct effect (10,11,12). As with normal subjects, COPD patients also show that ventilatory parameters during exercise are most closely related to dyspnea (13,14). Assuming that an increased elimination of carbon dioxide continues during recovery from exercise in these patients then carbon dioxide could be the predominant stimulus to ventilation and dyspnea during recovery (15). This could explain why oxygen supplementation is not effective in relieving dyspnea.

Another important consideration in COPD patients is the possibility of muscle fatigue during and following exhausting exercise. Respiratory muscles, as well as other skeletal muscles, exhibit both high- and low-frequency fatigue (16). Both types of fatigue would be expected to last longer than the duration of this study protocol and could have contributed to the trend towards a shorter endurance time with the second and third tests.

Importantly, from a clinical standpoint, although patients feel recovered and prepared to exercise again between 20 and 30 min after exhaustive walking exercise, the increase in recovery time we have demonstrated implies that physiological recovery is not complete. We also conclude that oxygen administration during exercise recovery

in COPD patients has no effect on reducing dyspnea recovery time and thus should not be recommended as intermittent treatment for post-exercise dyspnea in these patients unless specifically given to correct hypoxemia. These conclusions have clinical relevance for patients involved in exercise rehabilitation for COPD.

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